

(FILE 'HOME' ENTERED AT 09:19:42 ON 24 SEP 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS' ENTERED AT 09:19:53 ON 24 SEP 2002
L1 517 S (TRANSFER? OR TRANSFORM? OR TRANSFECT?) AND (NERVE CELL OR
NE
L2 138 S L1 AND PD<1999

L2 ANSWER 2 OF 138 CAPLUS COPYRIGHT 2002 ACS

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TITLE: Functional redundancy of acetylcholinesterase and
neuroligin in mammalian neuritogenesis

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AB Accumulated evidence attributes noncatalytic morphogenic activity(ies) to
acetylcholinesterase (AChE). Despite sequence homologies, functional
overlaps between AChE and catalytically inactive AChE-like cell surface
adhesion proteins have been demonstrated only for the Drosophila protein
neurotactin. Furthermore, no mechanism had been proposed to enable

signal

transduction by AChE, an extracellular enzyme. Here, we report impaired
neurite outgrowth and loss of neuroligin 1.alpha. mRNA under
antisense suppression of AChE in PC12 cells (AS-ACHE cells).

Neurite growth was partially rescued by addn. of recombinant AChE to the
solid substrate or by **transfection** with various catalytically
active and inactive AChE variants. Moreover, overexpression of the
homologous neuroligin 1 ligand, neuroligin-1, restored both neurite
extension and expression of neuroligin 1.alpha.. Differential PCR display
revealed expression of a novel gene, nitzin, in AS-ACHE cells. Nitzin
displays 42% homol. to the band 4.1 protein superfamily capable of

linking

integral membrane proteins to the cytoskeleton. Nitzin mRNA is high
throughout the developing nervous system, is partially colocalized with
AChE, and increases in rescued AS-ACHE cells. Our findings demonstrate
redundant neurite growth-promoting activities for AChE and neuroligin and
implicate interactions of AChE-like proteins and neuroexins as potential
mediators of cytoarchitectural changes supporting neuritogenesis.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR
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